EDITORIALS

Health Systems Agencies and Local Physicians

IT IS SAFE to assume that relatively few physicians and less of the public are more than vaguely aware of the National Health Planning and Resources Development Act of 1974 (PL 93-641) or of the proposed Health Planning and Resources Development Amendments of 1978 (HR 10460) now being considered by the Congress. Everyday patient care and professional practice will be profoundly influenced by the way this law is implemented and its reach no doubt extended. If the federal strategy (to use a federal term) succeeds, the local Health Systems Agencies (HSA's) will have a great deal to say about local planning for health care and the expenditure of federal funds for health in their area.

PL 93-641 grew out of three former federal programs and combined and strengthened them. These were Comprehensive Health Planning, Regional Medical Programs and the Hill-Burton Act. PL 93-641 divides all of the United States into health service areas (also HSA's). Within each area it sets up a Health Systems Agency (HSA). The local HSA is a regional or area agency which may be a private nonprofit corporation, a unit of local government, or part of a public multipurpose planning agency. Each HSA has a fulltime staff, paid by the federal government, who collect and analyze data and make recommendations to the HSA governing body. An HSA executive committee or governing body is made up of 20 to 30 members. Consumers must be in the majority. In addition several categories of "providers" must be represented in the nonconsumer portion. It works out that the slots for practicing physicians are quite limited, perhaps one or two on each HSA governing body. The same mix applies to all the HSA councils and committees as well.

The local HSA's are now in various stages of development throughout the nation. At the same time something of a four-way power struggle seems to be going on within government. The Congress perceives the power to lie in the consumer-dominated local HSA's, with federal guidelines to help them. The federal bureaucracy in the Department of Health, Education, and Welfare, responsible for developing the regulations, sees the federal guidelines more as federal mandates to be followed by the local HSA's. The state governments under the law have a role in coordinating statewide HSA activities and steering HSA's toward statewide goals, and they are presently jockeying with the federal government for the power to do this. And lastly, many local governmental authorities are seeking as much dominance and control of local HSA's as possible. So once again we seem to be embarked upon a massive, complicated and costly federal health program which is based upon a theoretical hypothesis which has never been tested and may or may not ever work. The potential for havoc is great, but there is also potential for good, for a more rational approach to health care at the local level, with some coordination at the state level and some help and guidance from the national level.

The omission, almost to the point of exclusion, of a significant presence of practicing physicians at each level of planning and decision making under this law seems to be a flaw which may prove serious and could even be fatal. There are great expectations of the conventional wisdom of the consumer and of the knowledge and expertise of other "providers" of health care. Yet it is the physician who in many ways is the most knowledgeable person of all about much of what is involved, and physicians are in a position to give expert counsel and advice which may or may not be sought, or listened to.

It is worth emphasizing that the HSA's may actually be ushering in a new period of participatory democracy in health care. This may prove to be the wave of the future. At present it is an experi-

ment with little accumulated experience with how it will work or even if it can be made to work. Physicians have been assigned a peripheral rather than a central role. But if this is indeed to be the wave of the future for health care in this nation, physicians had best prepare to adapt; that is, find ways how to participate effectively in the deliberations of the HSA's at the various levels. This will be a new dimension of medical practice which will require new approaches and new applications of what may be old skills. Since in a very real sense the HSA's will have health care problems, the aim will be for local physicians to participate in these democratic processes, especially at the local level, in much the same way they guide, teach, advise and help any of their patients who have health care problems. And, as is so often the case in other forms of medical practice, this will often be wearing, time consuming and frustrating. But it is essential that knowledgeable practicing physicians become involved in these matters of health and health care. Those who do this and do it successfully will often be rendering service to their profession and to the public well above and beyond the traditional call of duty. In retrospect they may even prove to have been the medical heroes of this moment in the evolution of health care in this nation.

—MSMW

Diagnosing Megaloblastic Anemias

IN THIS ISSUE Carmel reviews the laboratory diagnosis of megaloblastic anemias, emphasizing well-established, reasonably accessible laboratory tests. Another excellent review of the diagnostic approach to megaloblastic anemia is that of Chanarin,[1] published in an issue of *Clinical Haematology* devoted entirely to the megaloblastic anemias. Like Chanarin, Carmel stresses the importance of an elevated mean corpuscular volume (MCV) (that is, macrocytosis) in "tipping off" the diagnosis of megaloblastosis. An important fact which should also be mentioned is that the megaloblastic red cell is not only large but also oval; that is, it is a macroovalocyte. Its oval character distinguishes it from those large red cells that occur in hypothyroidism, in aplastic anemia, in liver disease not associated with vitamin deficiency and in various hemolytic anemias.[2]

Carmel notes that the question is unsettled as to whether subtle vitamin deficiency may explain the neutrophil hypersegmentation which has been reported in iron deficiency. We believe this question may have finally been laid to rest by our recent findings[3,4] that almost invariably there is underlying folic acid deficiency in iron deficient patients with neutrophil hypersegmentation.

A particularly interesting very recent finding is the report by Clarkson and Mockridge at the American Society of Hematology Annual Meeting in San Diego in December 1977[5] that one of the earliest events in the development of folate deficiency is a reticulocytopenia and a macroreticulocytosis which is present before the mature red cells become large, and which is accompanied by a low serum folate level, but occurs before the mean red cell folate becomes low.

Carmel notes that the white cell changes in so-called erythroleukemia, more accurately described as DiGuglielmo syndrome or preleukemia, tend to be less classical. Actually, it is a striking fact that in this syndrome there may tend to be hyposegmentation rather than hypersegmentation.[2] This seems to be analagous to the pseudo-Pelger-Huet anomoly which occurs in about 10 percent of patients with myelogenous leukemia at one time or another during their clinical progression.

The Carmel review of serum and red cell folate indicates that much depends on how one chooses to define deficiency. Perhaps it would be clarifying to quote verbatim from conclusion number two of the Proceedings of the Workshop on Human Folate Requirements, published in 1977:[6]

> What constitutes appropriate assessment of nutritional status with regard to folate? Answer: Measurement of serum plus red cell folate appears to be the most practical approach. Current evidence suggests that, when serum and red cell folate are both low, other more direct indices of folate deficiency will be present (i.e., a low liver folate and a bone marrow "dU suppression" test demonstrating subnormal DNA synthesis by bone marrow cells correctable by adding folate or methylfolate *in vitro*). Measurement of tissue folate (i.e., red cell or liver folate) alone is not adequate because deficiency of vitamin B_{12} results in low tissue folate (but not low serum folate). Low serum folate alone is not adequate as a test for folate deficiency because it is too sensitive; i.e., serum folate is low after only three weeks of folate deprivation, which is months prior to exhaustion of tissue folate stores and development of biochemical folate deficiency.

As Carmel notes, in the first few days after folate therapy, red cell folate is still low and it still can be used to determine that folate deficiency was present. More recent studies indicate